D-113

DATA ASSESSMENT REPORT

Technical Directive: 05
Technical Directive Title: Reilly Tar Technical Support

Technical Directive Report

by

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for

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July, 1982

INTRODUCTION

The complaint of the United States Government et al. against the Reilly Tar Co. alleges that chemicals and combinations thereof from the operation of a coal tar distillation facility which included the production of creosote and subsequent use of these materials to treat wood products were handled, stored, used and disposed of in a manner so that the surrounding areas were heavily contaminated and these chemicals now are present in water supplies in levels that pose serious potential health effects.

Approaches to establishing the complaint:

1) Analysis of ground waters for chemicals found in coal tar and its distillation products which are likely to pose a hazard to human health.

2) Geological and topographical analysis of the plant and surrounding area to demonstrate that contaminants from the facility could be the source of well water pollution.

3) Presentation of major toxicological information regarding the demonstrated adverse health effects of coal tar and certain distillation products.

4) Correlation of the levels of chemical contaminants with potential toxicological effects to assess the risk to human health.

1. Analysis of ground waters for chemicals found in coal tar and its distillation products which are likely to pose a hazard to human health.

The paper providing the description of the process at Reilly Tar is particularly revealing because this appears to have been a typical coal tar plant and wood treating operation with documentation of a sloppy operation both occupationally and environmentally. (I have submitted separately an example of a preliminary report regarding similar types of wood treating operations (Todd, et al., 1978)).

If one examines the literature regarding the composition of coal tar (coke oven) and one of its fractions, creosote, the case for the excessive contamination by toxic (and/or carcinogenic) compounds can be stronger. The work of Lijinsky (1963) and Lao, et al. (1975) provides guidance in the selection of additional PAHs. In addition, an effort should be made to search other sources, e.g. White (1975), for papers providing clues as to other compounds, e.g. β-naphthylamine. Actually the analysis of W13 for amines and heterocyclic compounds is important information in establishing the presence of compounds found in coal tar in the wells and making the point that coal tar contains numerous potentially toxic compounds.

Cocarcinogenic compounds in addition to those listed (pyrene, fluoranthene benzo(g,h,i)perylene) are worth pursuing. Phenolic compounds and more specifically catechol and pyrogallol are important as well as alkanes, alkylbenzenes and alkylnaphthalenes (Horton, 1957, Salaman and Roe, 1964, Slaga et al., 1980).

2. Geological and topographical analysis of the plant and surrounding area to demonstrate that contaminants from the facility could be the source of well water pollution.

This aspect appears to be the most thoroughly documented and certainly is important in the trail of events.

3. Presentation of major toxicological information regarding the demonstrated adverse health effects of coal tar and certain distillation products.

Ample evidence of the carcinogenic potency of coal tar exists in the open scientific literature. The first experimentally induced tumors were caused by applying coal tar to the skin of rabbit ears (Yamagiwa and Ichikawa, 1915). The pure chemical carcinogen, benzo(a)pyrene was isolated from coal tar in 1933 (Cook, 1933). Since that time coal tar (coke oven tar) and several of the carcinogenic and cocarcinogenic compounds contained therein have been intensely investigated.

A summary of the evidence of the carcinogenicity of benzo(a)pyrene in multiple species including the increased susceptibility of very young animals, and for a variety of target organs is described in an IARC Monograph (1973). In addition a variety of other chemicals have been identified either in coal tar or in fugitive coke oven emissions which, when captured in processing, form coal tar. Many are carcinogenic and include benz(a)anthracene, dibenzo(a,h)anthracene, etc. (See Table 1.).

Many investigations have used distillate fractions of coal tar in experiments to determine where the major carcinogenic activity is concentrated and what components contribute to its potency. The documentation for the hazards associated with exposure to coal tar are extensive and the summary from an IARC Monograph (1973) is attached.

Specific experiments and scientific information regarding the hazards associated with creosote appear in the occupational health literature. To summarize, creosote contains creosol, guaiacol, phenol, pyrol, pyridine and other aromatic compounds and the toxic effects reflect the constituent chemicals. The main cause of death after acute exposures is usually cardiovascular collapse. Fatalities have occurred 14-36 hours after ingestion of about 7 g. in adults or 1-2 g. in children. During episodes of self-medication, absorption from the gastroenteric tract induced disturbances of vision, digestion, hypertension and general cardiovascular collapse (Deichmann and Keplinger, 1963).

Contact with the skin may cause burning, itching, vesiculation, gangrene and is in some instances cancer. Photosensitization may also occur.

The evidence for carcinogenicity has been documented and is probably due mainly to compounds other than benzo(a)pyrene (Deichmann and Keplinger, 1963).

4. Correlation of the levels of chemical contaminants with potential toxicological effects to assess the risk to human health.

The most important aspect of this approach to providing evidence of a health

hazard is to rely on standards and criteria of performance already assued by USEPA (1980) and International Agencies. Factors, such as, PAHs from other sources and consumption levels of various foods have already been considered in these Water Quality Criteria and it makes little sense to reinvent them.

However the fact that the contaminants from coal tar include PAH and other toxic chemicals leads to the possibility of additive effects which may be stressed (See Table 2).

In an effort to provide emphasis concerning the potential toxicity of the contaminants it would be useful to prepare concentrates of water samples from various wells and submit them for short-term bioassay for their mutagenic potential and as a screen to predict carcinogenic potential (deSerres et al., 1981). Evidence of the mutagenic potential of chemicals actually in the wells would be impressive.

CONCLUSIONS

Evidence of a substantial hazard exists but the case can be made stronger by collecting some additional information in an organized, deliberate manner, specifically:

1- Analyze for additional potential contaminants that are carcinogenic and/or cocarcinogenic basing the selection on known constituents of coal tar (or coke oven emissions).*

2- Prepare more documentation of the toxic effects of coal tar (use this report as a basis for a bibliography), but also seek from NIOSH and EPA updates they may have prepared.

3- Continue to seek correlations or bits of data that may be supportive in preparing the case during a "brainstorming session". E.g. is it worthwhile to have a backup document on the increased susceptibility of young animals to carcinogen administration? Should a listing of the literature regarding possible associations or correlations between water supply and cancer be prepared? etc.

*It is not always clear that the paper trail for the samples and their analyses is adequate for the court so that any additional sampling (unless exploratory) should provide this documentation.

TABLE 1

CHEMICAL	OCCURRENCE	EVIDENCE FOR CARCINOGENIC ACTIVITY	
Benz(a)anthracene	Coal tar pitch (Wallcave, et al., 1971) Coal tar (Lijinsky, et al., 1963) Creosote (Lijinsky, et al., 1963)	+	Numerous references in IARC, pp.52-57.
Benzo(b)fluoranthene	Coal tar (Kruber & Oberkobusch, 1952)	+	A few references in IARC, p.74.
Benzo(j)fluoranthene	Coal tar (Kruber, et al., 1953) (Lijinsky, et al., 1963) Creosote oil (Lijinsky, et al., 1963)	+	Wynder & Hoffmann, 1959.
Benzo(a)pyrene	Coal tar & coal tar pitch (Lijinsky, et al., 1963) Creosote oil (Lijinsky, et al., 1963)	+	Numerous references in IARC, pp.102-114.
Benzo(e)pyrene	Coal tar (Lijinsky, et al., 1963) Coal tar pitch (Wallcave, et al., 1971) Creosote (Lijinsky, et al., 1963)	weak	A few references in IARC, p.146.
Chrysene	Coal tar (Lijinsky, et al., 1963) Creosote (Lijinsky, et al., 1963) Coal tar pitch (Wallcave, et al., 1971)	weak	A few references in IARC, pp.165-167.
Dibenz(a,h)anthracene	Coal tar (Lijinsky, et al., 1963)	+	Numerous references in IARC, pp.183-187.
Dibenz(a,i)pyrene	Coal tar (Schoental, 1957)	+	Numerous references in IARC, pp.217-219.
Indeno(1,2,3-cd)pyrene	Coal tar (Lang, et al., 1959) Coal tar pitch (Wallcave, et al., 1971)	+	Several references in IARC, pp.232-233.
Benz(c)acridine	Coal tar pitch (Kruber, 1941)	+	Few references in IARC, pp.243-244.

TABLE 2

CHEMICALS	TARGET ORGAN	REFERENCE
Benzo(a)pyrene (oral) + Nitrosamines (Mice)	Increased lung and stomach tumors beyond what is found with nitrosamine alone at the same dosage.	Schoental, 1963
Diethylnitrosamine + 4-dimethylamino-azobenzene (rats)	Same incidence of liver cancer found when only 66% of entire dose of carcinogen applied.	Schmahl, et al., 1963
4-dimethylamino-azobenzene + 3-methylcholanthrene	Increased induction of liver cancer over DAB alone (ordinarily 3-MC does not induce liver cancer).	Odashima, 1959
7,12-dimethylbenz(a)-anthracene (artificial diverticulum) + NN'-(2,7 Fluorenylene)-Bisacetamide (oral) (Fisher rats)	High incidence of tumors not found when either of the two compounds was administered alone (i.e. altered target sites).	Odashima, 1968.

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2.1 Human data

Butlin (1892c) described skin cancer among workers in the coal-tar and pitch industry. Largely through publications by Henry (1946, 1947) it was realized that the differing composition of tar products was related to the frequency of skin cancer among coal-tar workers. In 1907, the Workman's Compensation Act in England recognized officially that cutaneous epitheliomas could be caused by pitch or tarry substances. Ross (1948) classified the products obtained on distillation of coal-tar as shown in Table II:

TABLE II

Temperature	Compounds Produced
- 170°C	light oils; benzene, toluene, xylene
170° - 230°C	middle oils; phenols, cresols, naphthalene
230° - 270°C	creosote oils, tar oils
270° - 400°C	anthracene oils
	pitch residue

Exposures to pitch occur not only among coal-tar workers, but also among optical lens grinders, electrical equipment workers and wharfmen, cable layers, net fixers and fabric proofers (Jenkins, 1948). Exposure to creosote oil occurs among brick and tile workers, as well as among timber-proofers (Henry, 1946).

In contrast with cancer occurring in chimney sweeps, occupational exposure to tar and pitch affects predominantly skin sites other than the scrotum. From 1920-45, pitch or tar was deemed responsible for 2229 of 3753 notified industrial skin cancers in Great Britain (Henry, 1946, 1947); while during the decade 1946-55, 2041 new cases of occupational skin cancer were notified in Great Britain, of which 1053 were attributed to tar and pitch (Bogovski, 1960).

The majority of skin cancers in tar workers have been reported from England, but other reports have come from USA (Heller, 1930), Holland (de Vries, 1928), Germany (Volkmann, 1875) and France (Manouvriez, 1876).

Human skin cancers have been described after exposures to creosote oil (O'Donovan, 1928; Cookson, 1924; Lenson, 1956) and to anthracene oil (Bridge & Henry, 1928; O'Donovan, 1921).

Kennaway & Kennaway (1947) described an increased frequency of lung cancer among workers exposed to coal gas and tar. Doll (1952) found that among 2071 male pensioners of a London gas company, the number of deaths from lung cancers was approximately double that expected for male inhabitants of London of the same age (25 deaths observed vs 13.8 expected). The investigation concerned a multiplicity of occupations within the company, and it is probable that the risk was substantially greater for men most closely concerned with gas production.

In a further study, Doll et al. (1972) examined the mortality experience of groups of gas-workers in Great Britain over a period of 12 years. Among those categorized as coal carbonizing process workers, they found a significant excess of deaths from lung cancer (3.82 vs 2.13 per 100 000 per year); the rates for bladder cancer (0.40 vs 0.17) and cancer of skin and scrotum (0.12 vs 0.02) were also significantly higher than the national rates. Work as topman appeared to be particularly hazardous. The risk for lung cancer could not be related to any particular type of retort house. Among the by-products workers, there was no substantial evidence of any specific occupational hazard.

In Japan, 21 men exposed to coal-tar fumes in generator-gas plants developed lung cancer in a six-year period, a seeming excess over normal expectation (Kuroda & Kawahata, 1936). Kawai et al. (1967) observed that the longer the exposure the greater was the mortality from lung cancer, beginning ten years after exposure to coal-tar fumes.

In a British study of retort houses, certain areas were found to have 3 $\mu g/m^3$ benzo(a)pyrene, which was very much higher than the mean annual level for the city of London, away from the traffic, up to 1965. In the air above the retorts in the old horizontal retort houses where the processing

temperature was 800 to 1000°C, the concentration of benzo(a)-pyrene was over 200 µg/m^3. Such high levels were not found in vertical retort houses where the processing temperature was only 400 to 500°C (Lawther et al., 1965).

In a study of 58 528 employees of an American steelworks, Lloyd (1971) found that the mortality from respiratory cancer among coke plant workers was double that for steel workers as a whole. The excess was greater among those who had been employed directly at the ovens, particularly at the top of the ovens, among whom 15 lung cancer deaths occurred versus 1.5 expected.

Emissions from coke-ovens in the United States were analyzed for polycyclic aromatic hydrocarbons in 20 coke plants, and although their levels varied in each plant, similar ratios between benzo(a)pyrene, chrysene and benz(a)anthracene were found (Smith, 1970). In a report on a legal action involving one worker who developed lung cancer after working with tar, it was estimated that he could have inhaled 320 µg benzo(a)-pyrene per hour. The tar contained 3% benzo(a)pyrene and gave off vapours at 300°C containing 4.4% benzo(a)pyrene (Bonnet, 1962).

2.2 Animal data

Crude coal-tar was first shown to be carcinogenic experimentally by Yamagiwa & Ichikawa (1915) who painted it on rabbits' ears for several months; and later Tsutsui (1918) and Murray (1921) produced similar results on the skin of mice. Coal-tar pitch and anthracene oil, fractions of coal-tar, were also shown to be carcinogenic (Kennaway, 1925); Bonser (1932) produced skin cancers by administration of blast furnace tar. Twort & Fulton (1930) demonstrated that the carcinogenic potency of tars prepared at temperatures of 500°C, 600°C and 750°C increased materially with the temperature of preparation.

Lijinsky et al. (1957) have found commercial creosote oils to be highly carcinogenic. They contained 2.75 g/l benz(a)-anthracene, which is carcinogenic in the mouse (see monograph), and 1.27 g/l chrysene, a carcinogen for the mouse skin (see monograph). Benzo(a)pyrene and other pentacyclic PAH were present at much lower concentrations, i.e., about 50 mg/l.

2.3 Chemical composition

In an attempt to identify the responsible agent, carcinogenic coal-tar was fractionated and the aromatic fraction was found to be active. It possessed fluorescence, which led to the isolation and identification of benz(a)anthracene, a PAH with a characteristic fluorescence spectrum. On the basis of these findings, Kennaway & Hieger (1930), utilizing fluorescence spectroscopy, succeeded in isolating benzo(a)pyrene, which later proved to be carcinogenic, from coal-tar. With improved techniques, other PAH were isolated, among which dibenzo(a,h)-pyrene, dibenzo(a,i)pyrene, benzo(b)fluoranthene and dibenz(a,h)-anthracene (Badger, 1962) were also shown to be carcinogenic constitutents.

Kennaway (1925) manufactured carcinogenic tars synthetically from a variety of sources such as isoprene, acetylene, skin, yeasts and cholesterol, and showed that carcinogenicity increased with the temperature involved in the distillation of tars.